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Alpha-stat acid-base regulation during cardiopulmonary bypass

To the Editor:

I read with interest the recent study by Patel and associates,[1] particularly their opening comments that the issue of differing acid-base regulation "has not been tested in a relatively large trial of patients having CABG [coronary artery bypass grafting] alone." This is erroneous. Their lack of referral to a previous study by my colleagues and myself[2] is surprising considering that it was published in this same journal just 1 year previously. Their omission is all the more surprising because their results essentially confirm our observations that 2 months after the operation the incidence of cognitive dysfunction is reduced in patients having CABG who undergo cardiopulmonary bypass for longer than 90 minutes using alpha-stat management. It should be noted that the population they operated on were significantly younger than those we reported, the upper limit of their 95% confidence interval for age being less than the mean age of our population. Fundamentally, however, given differences in patient age and type of cognitive testing performed, the results of these two studies are quite comparable.

I am also puzzled by the authors statement that "there was a significantly greater reduction in $CMRO_2$ [cerebral metabolic rate for oxygen] in the pH-stat group during hypothermia." It is certainly not clear either from Fig. 3 or from the discussion on cerebral metabolism that, in fact, there was any significant difference in $CMRO_2$ between the alpha-stat and the pH-stat groups. What was the statistical strength of this observation? In a previous study, we[3] did not find any significant difference in $CMRO_2$ between groups despite cerebral blood flow (CBF) that was more than 50% lower in the alpha-stat group.

With respect to the absolute values of CBF and $CMRO_2$ that Dr. Patel's group measured, they do appear to be significantly higher than those same values that we had reported during cardiopulmonary bypass.[3] Although all of the factors mentioned by the authors, specifically, differences in perfusion technique and pressure management, can influence cerebral hemodynamics, it should also be borne in mind that anesthetics can have a significant influence on both CBF and $CMRO_2$. Accordingly, the patients that we had previously reported[3] received significantly higher doses of narcotic (fentanyl 0.1 mg/kg and a high dose of diazepam, 0.5 mg/kg), whereas their patients received 2 to 4 mg midazolam and 0.01 to 0.02 mg/kg fentanyl. I believe that when these fundamental differences in anesthetic management are taken into account, their CBF physiology measurements and those that we previously reported will be increasingly compatible.

Overall, the authors are to be congratulated for this study combining intraoperative cerebral physiology and postoperative cognitive performance.

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Reply to the Editor:

My coauthors and I would like to thank Professor Murkin for his comments relating to our article.[1] He neatly combines complimentary comments with a slight admonition relating to our unfortunate error in failing to cite his excellent study,[2] published in this Journal in August 1995, in which he addresses the issue of cognitive dysfunction and the effects of acid-base regulation during cardiopulmonary bypass (CPB) in patients undergoing coronary artery bypass grafting (CABG). In our defense, we can only apologize for this omission and state that our original manuscript was submitted to the Journal at the beginning of August 1995; invariably, our copy of the Journal does not arrive in the United Kingdom until 1 to 2 months later. Additionally, Professor Murkin cites our work in his study (reference 18), attributing it to *The Annals of Thoracic Surgery* as "in press"; this, however, is erroneous because we had never submitted the study to this journal although it had been submitted some 18 months previously to *Anesthesiology!*

We agree that our results essentially confirm those described in Professor Murkin's article. We also observed a significant reduction in cognitive dysfunction in the alpha-stat managed group of patients (20% vs 49% in the pH-stat group) at 6 weeks after the operation; these data compare well with the respective values (27% vs 44%) in the study by Murkin and colleagues at 2 months after the operation. However, differences in cognitive dysfunction between the acid-base management regimens in Murkin's study were not observed when all patients were taken into account; the above differences were observed only in those patients with CPB durations in excess of 90 minutes. In our study, all patients were included; the number of patients with CPB durations greater than 90 minutes in our study groups was 10